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Peer Effects on Weight Status, Dietary Behaviour and Physical Activity among Adolescents in Europe: Findings from the I.Family Study

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SUMMARY

This study uses survey data from the I.Family Study to investigate the association between adolescent and peer overweight in a sample of adolescents aged 12–16 from six European countries. We find clear evidence of peer effects on body mass index, waist circumference and body fat, which are stronger among adolescents at the upper end of overweight distribution. We also provide evidence that both consumption of less healthy foods and time spent in leisure time physical activity and audio-visual media are positively associated with similar behaviours among friends. These observations may suggest that peer effects on adolescent overweight operate by influencing friends' behaviour patterns, especially unhealthy food consumption and physical (in)activity.

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I. INTRODUCTION

At a time when around 2.8 million annual deaths in the European Union (EU) result from the consequences of overweight and obesity and around 7% of national health budgets are spent each year on obesity-linked diseases (European Commission, 2014), the prevalence of paediatric overweight or obesity is of particular concern (Ahrens et al., 2014). In EU countries, approximately 22 million children and “tweens” (i.e., children aged 10–12 years) are considered overweight or obese, with the numbers growing by 400,000 annually (European Commission, 2013). According to 2010 estimates from the World Health Organization’s Childhood Obesity Surveillance Initiative (The World Health Organization Regional Office for Europe, 2010), around one in three children aged 6–9 years in the EU is overweight or obese. These overweight/obese children are expected to face an increased prevalence of chronic diseases such as cardiovascular disease, strokes, type 2 diabetes and a subset of cancers (Hill and Peters, 1998), as well as certain social and mental health risks (OECD, 2012).

One important aspect of obesity among children, adolescents and even adults is whether it is influenced by the behaviour patterns and/or diets of peers, a question addressed by much recent literature in economics and other disciplines (Asirvatham et al., 2014; Christakis and Fowler, 2007; Cohen-Cole and Fletcher, 2008; de la Haye et al., 2011a; Gwozdz et al., 2015; Halliday and Kwak, 2009; Larson et al., 2013; Nie et al., 2015; Trogdon et al., 2008). A greater understanding of potential peer effects on obesity could increase the efficacy of targeted policies and boost the potential benefits of interventions through the so-called social multiplier mechanism (Fletcher, 2011), i.e. policies could benefit from the externality inherent in peer effects. It is primarily this social multiplier effect that has been of interest to economists. Should such an effect exist, then it would amplify any shock that affects individual behaviour as the sum of the individual effects would then be enhanced by the peer effect related to the social interactions (Fortin and Yazbeck, 2015). Numerous public policies aimed at combating obesity (including restrictions on food marketing to children, food labelling, information campaigns, taxes and subsidies) would benefit from such an externality. Currently, there is much debate on public policies that influence prices of unhealthy foods, especially through taxation. As long as a certain individual behavior gives rise to a negative externality that may lead to obesity within a social network, it may be justified to introduce a tax on this behavior – and the magnitude of the tax would depend on the size of the peer effect (Fortina and Yazbeck, 2015).

A more general economic justification for analyzing peer effects is that the economic costs associated with obesity are considered to be very high (Tremmel et al., 2017). In a comprehensive study for Germany, Lehnert et al. (2015) estimate the direct and indirect costs of overweight and obesity to approximately €18 billion in 2008, which also represented a 70% increase in costs within six years when compared to a similar earlier study (Konnopka et al., 2011). This increase is directly related to the rising prevalence of obesity. An important characteristic of peer effects is that they not only propagate unhealthy behavior within a social network (and thereby increase obesity rates), peer effects can also change societal perceptions of an ideal body weight (Gwozdz et al., 2015). Such changing perceptions may give rise to a persistency of high obesity rates.

Unfortunately, the existing literature on peer effects in adolescence is strongly dominated by U.S. studies, and we need to be cautious in generalizing these across diverse cultures and institutional settings (Gwozdz et al., 2015). European studies are limited and the potential mechanisms through which peer effects operate on individual weight status remain largely underexplored. To begin addressing this gap, we use survey data from the I.Family Study to test for peer effects on body fatness in a sample of adolescents aged 12–16 in six European countries. Unlike Gwozdz et al. (2015), our study identifies peer effects based on unique information about individuals that adolescents specifically designate as their friends. Because such a proximal definition of peers probably operates by influencing diet behaviour and physical activity (Trogdon et al., 2008), we explore the underlying mechanisms of peer effects on adolescents' bodyweight using a rich set of measures that identify dietary patterns (e.g. the Youth Healthy Eating Index (YHEI), consumption frequency of less and more healthy foods and time spent on leisure time physical activity (PA) and audio-visual media (AVM)). We define less healthy foods as sugar sweetened beverages (SSBs), simple sugars, fatty foods, and fast foods eaten as meals contrasting these with healthy foods like vegetables, fruits and other fibre-rich foods.

The contribution of our analysis to the literature on peer effects is twofold: first, it is one of the few European studies that focuses on peer effects of obesity among adolescence – and the only one to our knowledge that addressed the possible mechanisms through which the peer effect works within this population group. Second, our study uses a collection of objective measures on obesity, which few studies have at their disposal. As we highlight in the next section, having such rich objective data is important in order to credibly identify peer effects.

Overall, our results identify an association between adolescents' and their peers' overweight irrespective of whether the measure is body mass index (BMI), waist circumference or body fat. Conditional quantile regressions show that this association is strongly at and above the conditional bodyweight distribution median, especially for BMI and body fat. Interestingly, however, although we find clear evidence of a positive association between adolescent consumption of less healthy foods and peer consumption of similar foods, we find no such association for the consumption of healthy foods. Furthermore, adolescents' time spent on both leisure time PA and AVM is positively correlated with the time spent on those activities by their friends. Taken together, these findings suggest that peer effects on adolescent overweight operate through shared patterns of behaviour, particularly unhealthy food consumption and PA behaviour.

The remainder of the paper is structured as follows: Section 2 reviews the relevant research, Section 3 documents the data and methodology, Section 4 reports the results and Section 5 concludes the paper.

II. PRIOR RESEARCH

Since Christakis and Fowler's (2007) seminal paper, quite a large body of literature has evolved that investigates peer effects (see Table 1). Yet although a broad body of literature exists on the relation between peer effects and individual bodyweight, this research is dominated by studies based on U.S. data (Asirvatham et al., 2014; Christakis and Fowler, 2007; Cohen-Cole and Fletcher, 2008; de la Haye et al., 2011a; Fortin and Yazbeck, 2015; Fowler and Christakis, 2008; Halliday and Kwak, 2009; Larson et al., 2013; Leatherdale and Papadakis, 2009; O'Malley et al., 2014; Renna et al., 2008; Trogon et al., 2008; Valente et al., 2009; Yang and Huang, 2013). We are aware of only three recent studies that examine this topic in Europe (Gwozdz et al., 2015; Mora and Gil, 2013; Quinto Romani, 2014). Mora and Gil (2013) use data from a sample of secondary school students in Catalonia, Spain. They identify a positive and significant causal relation between adolescent BMI and friends' average BMI and also find that these peer effects are stronger than those reported for the U.S. Quinto Romani (2014) draws on a longitudinal data set from state schools in Aalborg, Denmark, and demonstrates that a targeted health intervention not only has a beneficial effect on the BMI of the individuals involved but also on that of peers not exposed to the intervention. This suggests peer health spill-over effects occur in a school setting. Gwozdz et al. (2015) use data from IDEFICS ("Identification and prevention of Dietary- and lifestyle-induced health Effects In Children

and infantS”) to show that although same-gender peer effects exist among the approximately 14,000 children aged 2–9 from 16 regions of 8 European countries, they differ by both region and the measure of overweight employed. For instance, peer effects are stronger in the more collective regions of Spain, Italy and Cyprus.

Table 1
Summary of studies of peer effects on individual bodyweight

Authors (year)	Data source	Country	Targets	Peer definition	Methods	Outcomes
Christakis and Fowler (2007)	Framingham Health Study	U.S.	Adults	Self-nominated friends, siblings, spouse, and neighbors	LLM	Positive
Cohen-Cole and Fletcher (2008)	Add Health	U.S.	Adolescents	Self-nominated friends	OLS Logit	Positive
Renna et al. (2008)	Add Health	U.S.	Adolescents	Self-nominated friends	OLS/IV	Positive
Trogdon et al. (2008)	Add Health	U.S.	Adolescents	Self-nominated friends; Students within the same grade	OLS/TSLS Probit/TSLS-Probit QR	Positive
Fowler and Christakis (2008)	Add Health	U.S.	Adolescents	Self-nominated friends	OLS/FE/MC	Positive
Halliday and Kwak (2009)	Add Health	U.S.	Adolescents	Self-nominated friends	OLS/Probit/FE	Positive
Valente et al. (2009)	In-school survey, Los Angeles	U.S.	Adolescents (11-15 yrs)	Self-nominated friends	RE-Logistic/ERGM	Positive

Yakusheva et al. (2011)	The study from a private Midwestern university	U.S.	Female freshmen	Roommates	NE	Negative
Yakusheva et al. (2014)	The study from two universities (private/public)	U.S.	First-year college students	Roommates	NE	Positive
Larson et al. (2013)	EAT 2010	U.S. (Minnesota)	Adolescents	Self-nominated friends	OLS/MR	Positive
Yang and Huang (2013)	Add Health	U.S.	Adolescents	Self-nominated friends	FE	Positive
Asirvatham et al. (2014)	Arkansas Center for Health Improvement	U.S. (Arkansas)	Children	Students within the same grade	OLS/FE/RE	Positive
De la Haye et al. (2011)	A public high-school survey in a major Australian city	Australia	Adolescents (12.3-15.6 yrs)	Self-nominated best friends	SAOMs	No effects
Leatherdale and Papadakis (2011)	SHAPES	Canada	Adolescents	Senior students (grades 11 and 12) within the same school	Logistic	Positive
Mora and Gil (2013)	A secondary-school student survey, Catalonia	Spain	Adolescents (14-18 yrs)	Self-nominated friends within the same classroom	OLS/GMM/LIML	Positive
Loh and Li (2013)	CHNS	Rural China	Adolescents (10-19 yrs)	Children in the same age group, level of school and community; Children in the same age group	OLS/2SLS/QR	Positive

				and community		
Gwozdz et al. (2015)	IDEFICS	Eight European countries	Children (2-9 yrs)	Children in the same age group, in the same school	OLS/FE	Positive
Quinto Romani (2014)	A longitudinal data of schoolchildren	Aalborg, Denmark	Adolescents (11-13)	Children in the same school	DID	Positive
Nie et al. (2015)	CHNS	China	Children (3-9) and adolescents (10-18)	Children in the same age group and community	OLS/QR/GMM/LIML/FE	Positive
Fortin & Yazbeck (2015)	Add Health	U.S.	Adolescents	Self- nominated friends	OLS/NLS/GMM	Positive

Notes: Based on Nie et al. (2015). Add Health = the National Longitudinal Study of Adolescent Health; EAT = the Eating and Activity in Teens in 2010; and SHAPES = the School Health Action, Planning, and Evaluation System. IDEFICS = Identification and prevention of Dietary – and lifestyle – induced health Effects In Children and InfantS. The estimation methods are as follows: Logistic=logistic model; LLM=longitudinal logistic-regression model; Ologit=ordered logit model; Probit= probit model; TSLS= two stage least squared model; IV-Probit= instrumental variable probit model; RE-Logistic= random effects logistic model; ERGM= exponential random graph model; SAOMs=stochastic actor-oriented model; FE=fixed effects model; RE=random effects model; MC=Monte Carlo simulations; DID=difference-in-difference; QR=quantile regression model; NE=natural experiment method (using random roommate assignments); NLS= non-linear least squares; GMM= general method of moments model; LIML=limited information maximum likelihood model; and MR= mean regression.

Most such research, however, fails to explore the potential pathways of peer effects on individual bodyweight – that is, whether peer effects operate through dietary or physical activity patterns, or by other channels such as perceptions/norms of body weight. There is some evidence that peers can influence perceptions of an ideal body weight or composition. For instance, Ali et al. (2011b), using data from Wave II (1996) of the U.S. National Longitudinal Survey of Adolescent Health (NLSAH), suggest that adolescents aged 11–20 who are exposed to heavier peers and overweight/obese parents are more likely to underestimate their own weight status. Likewise, Maximova et al. (2008), using data from the Quebec Child and Adolescent Health and Social Survey, show not only that a higher parental and schoolmate BMI is linked with greater underestimation of weight status among children and adolescents but that overweight and obese youth are more likely to underestimate their own weight relative

to non-overweight peers. This latter is echoed by Gwozdz et al. (2015), whose analysis indicates that parental underestimation of their own children's weight go hand in hand with fatter peer groups among the children. Similarly, Blanchflower et al. (2009) demonstrate that self-perception of overweight is affected by an individual's BMI relative to a broadly defined peer group.¹ However, evidence as to whether and how peers may affect dietary behaviours and especially physical activity is scant (Salvy et al., 2012). One interesting exception is Fortin and Yazbeck's (2015) analysis of four waves from the Add Health survey of American adolescents in grades 7 through 12, which identifies positive (albeit small) peer effects on fast food consumption among adolescents within the same school friendship network.

Unfortunately, almost all the above studies use BMI as a measure of overweight, and the majority rely on self-reported measures (most notably, from the NLSAH), both of which are considered problematic. Many criticize the reliability of BMI as a proxy of individual fat on the basis of its inability to distinguish fat from muscle, bone and other lean body mass (Barlow, 2007; Burkhauser and Cawley, 2008; Gallagher et al., 1996; McCarthy et al., 2006; Romero-Corral et al., 2006; Wellens et al., 1996; Yusuf et al., 2005). In addition, epidemiologists disparage self-reported weight and height data because of the potential for reporting biases (Huybrechts et al., 2006; Shields et al., 2011).

This paper therefore contributes to the literature in two important respects: First, it analyses potential peer effects on a range of objective measures of body overweight. Second, it explores the specific mechanisms through which peers might influence adolescent body overweight by assessing peer effects on obesogenic behaviours (diet, physical activity and sedentary behaviours).

III. DATA AND METHODS

1. Survey and sample

Our data are taken from the I.Family Study, a 2013/2014 follow-up to the IDEFICS cohort and intervention study (Ahrens et al., 2017). I.Family covers not only the children from the original IDEFICS cohort but also their siblings and newly recruited children. It was designed to assess the interplay between complex lifestyle, social, behavioural and genetic factors and their

¹ Here relative BMI is measured as an individual's BMI divided by the averaged BMI from their country, age band and gender cell (Blanchflower et al., 2009).

impact on dietary habits and health outcomes, and one strength of this survey lies in its detailed information on body fatness. Trained field staff measured skinfolds, waist and hip circumference, bioelectrical impedance and ultrasonography as well as height and weight, giving us several measures of body composition. They also took venous blood and mouth mucosal cell DNA from particular sub-samples to collect biochemical and gene expression markers.

To compile our analytic sample of 12- to 16-year-olds from Cyprus, Estonia, Germany, Hungary, Italy, and Sweden, we selected only those subjects for whom detailed information is available on demographic, parental and household characteristics and on designated friends (Arvidsson et al., 2015). This leaves a final sample of 655 observations for BMI, 646 observations for waist circumference, and 646 observations for body fat.

2. Peers

Peers are identified based on an item in the written self-administered survey that asks respondents to provide the names and grade/class of up to 10 friends and indicate the closeness of the friendship (see Appendix A1). A second step then identifies friends who are also participating in the I.Family Study, after which all data are anonymized. By including only these participating friends in our sample, we ensure a rich data set of matching information.

3. Dependent variables

Weight status measures. Our analysis is based on three measures to derive level of overweight/overfatness measures: (i) BMI z -values calculated using International Obesity Task Force (IOTF) growth charts (Cole et al., 2000)², (ii) waist circumference z -values calculated based on IOTF growth charts, and (iii) body fat estimated by a composite measure developed using field-derived data on hip circumference, triceps skinfold and resistance (measured with bioelectrical impedance analysis), together with z -values for body fat based on IOTF growth charts. As in (Gwozdz et al., 2013), our choice of methods is determined largely by Bammann et al.'s (2013) validation of the obesity measures in the IDEFICS study.

Diet. Dietary patterns are measured by the self-administered food frequency questionnaire (FFQ) section of the Children's Eating Habits Questionnaire (CEHQ) (Arvidsson et al., 2015),

² We use the IOTF growth charts which is the common approach when using international data, i.e. data from several countries. The thresholds for classifying children are derived from a reference population. The IOTF thresholds are derived from body mass index data from six large, nationally representative, cross-sectional surveys from Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the United States (see Cole et al., 2000).

which asks adolescents about specific consumption frequencies of 59 food and beverage categories. The I.Family version of the CEHQ-FFQ is comparable to its previous version, a reproducible and validated instrument used during the IDEFICs study (Bel-Serrat et al., 2013; Huybrechts et al., 2011; Lanfer et al., 2011). In I.Family, all 59 items are based on the question, *“In the last month, how many times did you eat or drink the following food items?”* with response categories coded as follows: 1 = never/less than once a week, 2 = 1–3 times a week, 3 = 4–6 times a week, 4 = 1 time per day, 5 = 2 times a day, 6 = 3 times a day and 7 = 4 or more times a day. We recode these categories to capture weekly consumption and then use the category mid-point as a proxy for weekly consumption frequency (1 = 0, 2 = 2, 3 = 5, 4 = 7, 5 = 14, 6 = 21 and 7 = 30). We define unhealthy food consumption as the consumption of sugar-sweetened beverages (SSBs), simple sugar foods (sugar),³ fatty foods (fat)⁴ and fast foods eaten as meals. Healthy food consumption includes consumption of vegetables and fruits (vegetables)⁵ and other fibre rich foods (fibre).⁶ Comparability across countries was ensured in the original survey by the same foods and beverages being translated into national languages (Arvidsson et al., 2015). The CEHQ-FFQ measures fast food consumption based on the question, *“How many times do you consume a full meal alternative to a main meal (breakfast, lunch, dinner) in a fast food restaurant?”*, measured on a 5-point scale from 1 = never to 5 = 3 or more times a week. We calculate the other food consumption indicators by adding corresponding food categories from the CEHQ-FFQ.

As a further measure of healthy eating, we introduce the 100-point Youth Healthy Eating Index (YHEI) (Feskanich et al., 2004), on which a higher score indicates a healthier diet. Our data set enable to replicate 10 of the 13 original YHEI dimensions, seven designed to measure food consumption and three to identify food-related behavioural patterns, as follows: (1) whole grains (source of fibre, vitamins and minerals), (2) vegetables (source of vitamins and minerals), (3) fruits (source of vitamins), (4) dairy (source of calcium), (5) snack foods (unnecessary energy), (6) soda and drinks (unnecessary energy), and (7) margarine and butter (sources of fat), (8) fried foods outside home (high energy intake), (9) eat breakfast (indicator of healthy dietary patterns) and (10) dinner with the family (indicator of healthy dietary

³ The sugar indicator comprises fruit juices, sugar sweetened drinks, sweetened or sugar added breakfast cereals and muesli, sweetened and/or flavoured milk and yoghurt, sweet snacks and ice cream and jams and honey.

⁴ Fatty food consumption covers fried potatoes, fried fish, fried meat, fried or scrambled eggs.

⁵ The indicator vegetables and fruits refers to potatoes and other cooked vegetables, legumes, raw vegetables, and fresh fruits with and without added sugar.

⁶ Fibre consumption includes potatoes and other cooked vegetables, legumes, raw vegetables, fresh fruits with and without added sugar, wholemeal bread, pasta, noodles, rice and other cereals, nuts, seeds and dried fruits, as well as porridge, oatmeal, gruel, unsweetened cereals and plain muesli.

patterns). We calculate scores for each based on the criteria proposed by Feskanich et al. (Feskanich et al., 2004), and then sum all available scores for the 10 dimensions.

Physical activity and sedentary behaviours. We measured self-reported time spent outdoors in leisure time using the following question: “*How much time do you spend playing or ‘hanging out’ outdoors on a typical day in your leisure time?*”. This was reported separately for both weekday and weekend day and combined for our analytic measure into total amount of time (in hours) spent on leisure time PA per week. We also measured time spent in audio visual sedentary behaviours (audio visual media – AVM) by the questions: “*How much time do you spend watching TV shows, movies or music videos?*” Time (hours per week) spent either watching TV or using a personal computer/laptop (with/without Internet access) was the measure used in the analysis.

4. Control variables

Following the extant literature of peer effects on individual bodyweight (e.g. Gwozdz et al., 2015; Mora and Gil, 2013), we examined a series of control variables comprising characteristics of the individual adolescents, of his or her mother and family. The adolescent characteristics comprise six variables: age, sex, meal frequency (times a day), health-related quality of life (KINDL), number of I.Family Study friends and time spent with these friends. Sex is a dummy equal to 1 if the adolescent is male and 0 otherwise. Health-related quality of life is based on the Questionnaire for Measuring Health-Related Quality of Life in Children and Adolescents KINDL – an instrument that has been tested in 13 European countries and turned out to be cross-culturally valid (Ravens-Sieberer et al., 2008). The instrument includes four dimensions: emotional wellbeing, self-esteem, parent relations and social contacts. The sum score of all items ranges from 0 to 64, with a higher score denoting a better quality of life (Bullinger et al., 2008). Time spent with friends is measured on a scale from 1 = “a lot of” time to 5 = “some” time. We also introduce four mother and family characteristic variables: mother’s age, occupational status, BMI and household income. Mother’s occupational status is measured on a 6-point scale of 1 = not employed, 2 = full time, 3 = part time ≥ 15 hours a week, 4 = part time ≤ 15 hours a week, 5 = on leave and 6 = in education. We recode this score as a dummy with “not employed” as the reference category. We similarly convert the household income scale of 1 = low level, 2 = low-medium level, 3 = medium level, 4 = medium-high level and 5 = high level to a dummy with “low level” as the reference group.

5. Estimation strategies

Ordinary least squares (OLS). To test for the existence of peer effects on adolescent's weight status based on the three z -scores for BMI, waist circumference and body fat, we estimate the following OLS model:

$$T_i = \beta_0 + \beta_1 P_i + \beta_2 X_i + \beta_3 M_i + \beta_4 F + \beta_5 C + \varepsilon_i \quad (1)$$

where T_i denotes the overweight measures of adolescent i , and P_i represents the corresponding average overweight measure of the adolescent's designated friends. X_i is a vector of adolescent i 's characteristics, and M_i is a vector of adolescent i 's mother's characteristics. F denotes the family characteristic in form of household income dummies, C is a country dummy, β_1 is the key coefficient of interest, and ε_i is the error term.

Multilevel mixed-effects generalized linear model (MMGLM). We then control for contextual effects such as shared environments by employing the following multilevel mixed-effects generalized linear model. MMGLM allows taking care of the data structure, i.e., individuals as first level and country as second-level random effect:

$$T = \beta X + \gamma Z + \varepsilon \quad (2)$$

where T is a column vector of adolescent's overweight, X is a matrix of the predictor variables for fixed effects and β is a column vector of the fixed-effects regression coefficients. Z is a matrix of random effects, γ is a vector of random effects (here, the random complement to the fixed-effects coefficient β), ε is a vector of error term and the $\beta X + \gamma Z$ term is the linear predictor. We employ a two-level random intercept model with country as the higher level and adolescents as the micro level.

Quantile regressions. Lastly, to assess whether mean peer overweight impacts differently across the distribution of individual weight status (conditional on control variables), we estimate the following quantile regression model at the 25th, 50th and 75th percentiles using the same specifications as in the OLS model:

$$T_i^q = \beta_1^q P_i + \beta_2^q X_i + \beta_3^q M_i + \beta_4^q F + \beta_5^q C \quad (3)$$

where q denotes different quantile levels, and β_1^q is the key coefficient of interest. It is worth emphasizing that, relative to mean-based regressions (e.g. OLS estimation), quantile regressions allows the peer effects to differ over the quantiles of individual bodyweight. Thus, quantile regressions allow us to detect whether obese individuals are more vulnerable to their peers.

IV. RESULTS

1. Descriptive statistics

As appendix Table A2 demonstrates, the average z -scores of the adolescents' BMI, waist circumference and body fat are 0.688, 0.932 and 0.584, respectively. The corresponding z -scores for their peers are 0.691, 0.937 and 0.567, respectively. The average age of all the adolescents is approximately 13 years, 45% of the sample is male, and the adolescents have an average 2.7 designated school friends. It is also worth noting that, on average, they spend about 14 hours a week on leisure time PA versus 24 hours using AVM. The adolescents' mothers have an average BMI of around 25 and 51% are full-time employed.

2. Peer effects and adolescent overweight

OLS and MMGLM estimates. The results for the models with and without controls are reported in Table 2.⁷ Column 1 shows that with only peer overweight controlled for, individual overweight significantly and positively correlates with average peer overweight, although magnitudes vary depending on the measurement used (BMI: 0.258; waist circumference: 0.218; body fat: 0.358). These coefficients remain uniformly significant and positive even after columns 2 and 3 introduce individual, mother and household controls and country dummies (Column 2 – OLS with controls: BMI: 0.100; waist circumference: 0.101; body fat: 0.148 and Column 3 – MMGLM: BMI: 0.111; waist circumference: 0.114; body fat: 0.158).⁸

⁷ As a robustness check, we also generate a weighted average of the friends' bodyweight through the frequency that each adolescent spends with his/her friends. The results (Table A3 in the Appendix) are quantitatively similar to those in Table 2.

⁸ We also introduce peer's averaged mother age, occupational status and BMI in order to control for contextual effects. The results (Table A4 in the Appendix) are quantitatively similar to those in Table 1. A similar strategy has been used in other related studies, for instance, Fortin and Yazbeck (2015) for the U.S and Mora and Gil (2013) for Spain.

Table 2
OLS/MMGLM estimates of peer effects on adolescent weight status

Variable	OLS	OLS	MMGLM
	(1)	(2)	(3)
<i>BMI (z-score)</i>			
Average peer BMI	0.258***	0.100*	0.111***
<i>SE</i>	(0.050)	(0.051)	(0.025)
95% <i>CI</i>	[0.160,0.357]	[0.001,0.200]	[0.063,0.160]
<i>N</i>	655	655	655
<i>R</i> ²	0.042	0.291	
<i>Waist circumference (z-score)</i>			
Average peer waist circumference	0.218***	0.101*	0.114***
<i>SE</i>	(0.050)	(0.048)	(0.013)
95% <i>CI</i>	[0.120,0.316]	[0.007,0.196]	[0.089,0.139]
<i>N</i>	646	646	646
<i>R</i> ²	0.029	0.245	
<i>Body fat (z-score)</i>			
Average peer body fat	0.358**	0.148**	0.158***
<i>SE</i>	(0.047)	(0.051)	(0.032)
95% <i>CI</i>	[0.265,0.450]	[0.047,0.249]	[0.095,0.222]
<i>N</i>	646	646	646
<i>R</i> ²	0.080	0.298	
Controls	No	Yes	Yes

Notes: The dependent variables are individual z-scores of BMI, waist circumference (WC) and body fat (BF) based on IOTF criteria. The controls are adolescent characteristics (age, gender, AVM consumption, meal frequency, health-related quality of life, number of friends and time spent together) and mother and family characteristics (age, occupation, BMI and household income). Dependent on regression type, we use country dummies as fixed or random effects. Robust standard errors are in parentheses (*SE*); 95% confidence intervals (*CI*) are in brackets.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

Conditional quantile regressions. As Table 3 illustrates, the coefficient of average peer overweight is significantly positive in the median and upper part of the distribution, especially for BMI and body fat. Regarding body fat, the coefficient of average peer overweight at the 75th percentile is stronger than that at the median part of the distribution (50th: 0.146 vs. 75th: 0.167). The results are consistent with those of Trogon et al. (2008) for the U.S and Nie et al. (2015) for China. With regard to waist circumference, only adolescents at the median distribution show a significant positive association (50th = 0.127). This observation of heterogeneous associations produced by different measures of overweight is in line with Burkhauser and Cawley's (2008) finding that different measures of obesity correlate differently with different outcomes of interest. The general impression one gets from Table 3 is that the peer effect is relatively weaker and insignificant in the lower ends of the body weight distributions. Taken at face value, these results would imply that peer weight is more influential

among adolescents with higher body weight and they would also provide an explanation for the rise in the right tail of body weight distributions. Policy measures that are aimed at overweight adolescents would thus be particularly effective, i.e., profit the most from the social multiplier effect. However, as pointed out by Trogon et al. (2008), another possible reason for this observation is that homophily (i.e., selection) might be higher at the upper end of the body distribution than in the lower end.

Table 3
Quantile estimates of peer effects on adolescent weight status

Variable	25%	50%	75%
	(1)	(2)	(3)
<i>BMI (z-score)</i>			
Average peer BMI	0.097	0.187**	0.133*
SE	(0.060)	(0.060)	(0.067)
95% CI	[-0.022,0.215]	[0.070,0.305]	[0.001,0.265]
N	655	655	655
Pseudo R ²	0.182	0.185	0.188
<i>Waist circumference (z-score)</i>			
Average peer WC	0.109	0.127*	0.102
SE	(0.067)	(0.059)	(0.064)
95% CI	[-0.384,0.094]	[-0.374,0.046]	[-0.483,-0.028]
N	646	646	646
Pseudo R ²	0.145	0.152	0.170
<i>Body fat (z-score)</i>			
Average peer BF	0.115	0.146*	0.167*
SE	(0.068)	(0.067)	(0.072)
95% CI	[-0.018,0.248]	[0.015,0.277]	[0.026,0.309]
N	646	646	646
Pseudo R ²	0.178	0.176	0.185

Notes: The dependent variables are individual z-scores of BMI, waist circumference (WC) and body fat (BF) based on IOTF criteria. The controls are adolescent characteristics (age, gender, AVM consumption, meal frequency, health-related quality of life, number of friends and time spent together) and mother and family characteristics (age, occupation, BMI and household income). Bootstrapped standard errors are in parentheses (SE); 95% confidence intervals (CI) are in brackets.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

3. Mechanisms

Table 4 reports the results for peer effects on adolescent (un)healthy dietary patterns and PA. Three observations are worth noting: First, in line with prior studies for the U.S. and Netherlands (Bevelander et al., 2012; Salvy et al., 2007a; Salvy et al., 2012; Salvy et al., 2007b), unhealthy food consumption by peers is consistently and positively associated with the adolescents' own unhealthy consumption even after controls are taken into account (Table 4, panel A, columns 1–3). We find no such association, however, between adolescent and peer

healthy food consumption. Likewise, although adolescent YHEI in the baseline model is positively and significantly correlated with peer YHEI (panel B, column 1), no such association exists once we take controls into account (Table 4, panel B, columns 2 and 3). Lastly, the results reveal significant positive correlations between the adolescents' leisure time PA and AVM consumption and those of their peers (Table 4, panel C, columns 1–3), which supports the notion that adolescents tend to befriend peers who engage in similar amounts of PA and continue to share similar patterns with their friends (de la Haye et al., 2011b).⁹

Table 4
OLS/MMGLM estimates of peer effects on adolescent diets and PA

Variables	OLS (1)	OLS (2)	MMGLM (3)
Panel A: Unhealthy diets			
<i>SSB consumption (times per week)</i>			
Average peer SSB consumption	0.197**	0.157*	0.168**
SE	(0.058)	(.060)	(.059)
95% CI	[0.082,0.312]	[0.038,0.275]	[0.053,0.282]
N	383	383	383
R ²	0.026	0.105	
<i>Sugar consumption (times per week)</i>			
Average peer sugar consumption	0.187**	0.171**	0.174**
SE	(0.054)	(0.054)	(.053)
95% CI	[0.082,0.293]	[0.065,0.278]	[0.071,0.277]
N	548	548	548
R ²	0.022	0.077	
<i>Fatty food consumption (times per week)</i>			
Average peer fat consumption	0.255**	0.218**	0.241**
SE	(0.050)	(0.051)	(0.049)
95% CI	[0.156,0.353]	[0.118,0.319]	[0.144,0.337]
N	548	548	548
R ²	0.045	0.130	
<i>Fast food consumed as meals</i>			
Average peer fast food consumption (meals)	0.201**	0.171**	0.180**
SE	(0.049)	(0.049)	(0.049)
95% CI	[0.104,0.298]	[0.074,0.267]	[0.084,0.275]
N	629	629	629
R ²	0.026	0.104	
Panel B: Healthy diets			
<i>Fibre- rich food consumption (times per week)</i>			
Average peer fibre consumption	0.104	0.057	0.069
SE	(0.054)	(0.054)	(0.053)
95% CI	[-0.002,0.210]	[-0.050,0.163]	[-0.035,0.174]
N	540	540	540
R ²	0.007	0.086	

⁹ We have also estimated the quantile regressions for peer effects on diets, physical activity and sedentary activity. The results indicate that, for sugar consumption, peer influence is stronger at the upper distribution of individual sugar consumption than that at the median (75th: 0.139 vs. 50th: 0.121). In addition, regarding leisure time PA, we find that peer influence is much stronger at the median than that at the 25th percentile (0.309 vs. 0.104). For other unhealthy diets, physical activity and sedentary activity, we cannot observe any heterogeneities among different percentiles. The results are available from the authors upon request.

<i>Vegetable/fruit consumption (times per week)</i>			
Average peer vegetable consumption	0.072	0.044	0.049
SE	(0.049)	(0.049)	(0.048)
95% CI	[-0.024,0.168]	[-0.053,0.141]	[-0.046,0.144]
N	589	589	589
R ²	0.004	0.063	
<i>YHEI (0-100)</i>			
Peer YHEI	0.201**	0.084	0.085
SE	(0.053)	(0.054)	(0.053)
95% CI	[0.097, 0.304]	[-0.022, 0.190]	[-0.020, 0.189]
N	641	641	641
R ²	0.022	0.126	
Panel C: PA and sedentary activity			
<i>Leisure time PA (hours per week)</i>			
Average peer PA	0.283**	0.262**	0.272**
SE	(0.053)	(0.052)	(0.054)
95% CI	[0.178,0.387]	[0.153,0.371]	[0.167,0.377]
N	466	466	466
R ²	0.058	0.110	
<i>AVM consumption (hours per week)</i>			
Average peer AVM consumption	0.238**	0.106*	0.119*
SE	(0.045)	(0.048)	(0.046)
95% CI	[0.149, 0.327]	[0.011, 0.200]	[0.028, 0.210]
N	610	610	610
R ²	0.043	0.167	
Controls	No	Yes	Yes

Notes: The dependent variables are consumption frequency of the various food types (SSBs, simple sugar foods, fatty foods, fast foods consumed as meals/snacks or fibre-rich foods, vegetables/fruits), as well as time spent on PA. Frequencies of fast food consumption as meals or snacks are coded as follows: 1 = never, 2 = once a month or less, 3 = several times a month, 4 = 1–2 times a week and 5 = ≥ 3 times a week. The controls are adolescent characteristics (age, gender, meal frequency, health-related quality of life, number of friends and time spent together) and mother and family characteristics (age, occupation, BMI, household income). Dependent on regression type, we use country dummies as fixed or random effects. Robust standard errors are in parentheses (*SE*); 95% confidence intervals (*CI*) are in brackets.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

4. A note on endogeneity

Despite wide recognition that inadequate control for peer-group endogeneity, shared environmental influences, and simultaneity can lead to the inflation of peer effects on weight gain (Fletcher, 2011), few studies sufficiently take all these issues into account. In particular, an individual's bodyweight might be correlated with that of a peer group stemming from an endogenous effect, contextual effect, correlated effect, or selection effect. The first denotes a direct influence of the peer group on the individual, whilst the second acknowledges that an individual's bodyweight could be affected by peer group characteristics other than bodyweight (Nie et al., 2015). The third represents that both the individual's and the peers' body weight may be affected by some unobservables (e.g., physical exercise at school), and the fourth recognizes that obese individuals may select friends that are themselves obese. As Trogon et

al. (2008) have emphasized, if a correlation between individual and peer-group obesity emanates from one of the last three effects, then interventions aimed at reducing obesity are less likely to lead to the oft-cited social spill-over effect. Furthermore, not adequately controlling for these effects may result in an overestimation of the peer effect (Fletcher, 2011).

Although the most common way to tackle this issue is by implementing an instrumental variable (IV) approach using the peers' average parental BMI as instruments (Nie et al., 2015; Renna et al., 2008; Trogon et al., 2008), the validity of doing so is largely dependent on the (critical) assumption that the selection of peers and indirectly of peers' parents is not correlated with an individual's BMI (Trogon et al., 2008). This assumption, however, is almost certainly false. We know that "friendships could also be selected on the basis of obesity status, with obese youths relatively likely to have obese parents. This strategy may also suffer from a second-order case of the reflection problem – friend's parents' weight may be affected by friend's weight which in turn may be affected by the respondent's weight" (Cawley and Ruhm, 2011, p. 136). Perhaps a more promising approach is the Mendelian randomization (MR) approach using genetic variants as instruments, mainly because genes are inherently randomized by a naturally occurring process, assigned at conception, not directly visible and thus unlikely to be related to other individuals (O'Malley et al., 2014; von Hinke et al., 2016). The MR approach is also advantageous because it measures genotypes with higher accuracy, identifies long-term exposure to outcomes of interest, and is immune to biases due to measurement errors (Haycock et al., 2016). Nonetheless, it is also subject to such shortcomings as identification problems related to biological mechanisms, genetic coinheritance and population stratification (von Hinke et al., 2016). In particular, evidence on the biological mechanisms through which the genetic variants may affect individual bodyweight is sparse. It could be that the genetic variants might be associated with other phenotypes and the instruments we use may thus be invalid if those genetic variants affect the outcome of interest directly (von Hinke et al., 2016). Regarding genetic coinheritance (so-called linkage disequilibrium, indicating the association between alleles at different loci within the population), "linkage disequilibrium can exist because alleles are physically close together and tend to be co-inherited, or because they occur together for reasons of population origin in subsections of an overall population and therefore demonstrate a statistical association within the overall population" (Davey Smith and Ebrahim, 2003, p. 5). In such a case, potential violation of the exclusion restriction largely rests on the functions of any co-inherited variants, and on whether those relate to the outcome of interest (von Hinke et al., 2011). Furthermore,

the allele frequencies of the single nucleotide polymorphism (SNP) – that explains the largest proportion of the variance – are known to differ by ethnic group (Frayling et al., 2007). The independence assumption might thus be violated when population stratification (due to ethnicity) exists.

Despite these drawbacks, and in order to take advantage of the rich genetic and parental background information provided by the I.Family Study while also acknowledging the inherent endogeneity in our previous models, we combine both these methods in our analysis. Given the evidence that the fat mass and obesity gene (*FTO*) and the melanocortin-4 receptor gene (*MC4R*) are strongly correlated with overweight and obesity (Lauria et al., 2012; Liu et al., 2013; Speliotes et al., 2010), we follow O'Malley et al. (2014) by employing them as instruments.¹⁰ As in von Hinke et al. (2016), we adopt an additive model to create an unweighted allelic score by summing up the number of obesity-risk alleles carried by each individual to improve the power of the instruments and alleviate weak IV problems. Specifically, the unweighted allelic score is calculated by summing the numbers of the homozygous adiposity-risk genotypes of *FTO* (rs9939609) (TT = 0, AT = 1, and AA = 2) and *MC4R* (rs17782313) (TT = 0, CT = 1, and CC = 2) for each of an adolescent's designated friends. As emphasized by von Hinke et al. (2016), controlling for covariates is particularly important in the presence of population stratification (as in our case) even though the MR approach relies on an unconditional independence assumption. We therefore include the same controls as in the above analysis but employ a two-step generalized method of moments (GMM), which is efficient in the presence of the heteroscedasticity observed here. We also use a limited information maximum likelihood (LIML) estimation, which is generally preferable when instruments are weak.

Using the traditional IV approach, we find no association between adolescent BMI and peer average BMI¹¹ (Table 5, approach 1, panel A) – even though peer parental BMI is significantly and positively associated with peer BMI in the first-stage estimates, regardless of whether the estimates are LIML or GMM (Table 5, approach 1, panel B). Likewise, using MR estimation shows that peer effects vanish when the unweighted allele score is used (Table 5, approach 2,

¹⁰ Put simply, *FTO* (rs9939609) and *MC4R* (rs17782313) are used to calculate the unweighted allele score, where the rs-number is an identification tag that uniquely identifies the polymorphism in the genome (von Hinke et al., 2016).

¹¹ When we apply the same MR strategies to waist circumference and body fat, we generally obtain that results are quantitatively similar to those in Table 4 (available from the author upon request).

panel B).¹² For MR estimation, we also perform the weak identification test to assess the validation of our IV. And the results based on the Wald F statistics suggests that our IV suffers from the issue of weak instrument. Therefore, we adopt the LIML instead of 2SLS. At first sight, these results may seem to call into question the very existence of peer effects. Given the substantial drawbacks of both approaches (especially in our setting), however, we believe they warrant no such conclusion. First, an IV approach based on peer background information could be problematic because it comes at the high price of increased measurement error and weaker instruments (Halliday and Kwak, 2009). Second, although the MR method, particularly using unweighted allele scores, might be able to solve the weak instrument and selection bias issues, the above-mentioned identification problems related to biological mechanisms, genetic coinheritance and population effects remain (von Hinke et al., 2016). Third, and most important in our case, the MR approach requires large sample sizes that encompass both genetic biomarkers and outcomes of interest; otherwise, genetic variants would merely suggest that, as in our case, the peer overweight was not varied enough to affect the adolescents' overweight (von Hinke et al., 2016). It would thus be an over-interpretation of (limited) genetic data to claim that there is no causal relation.

Finally, the same sources of endogeneity also exist for our analyses of PA and diets, yet finding appropriate instruments for these regressions is even harder than in the case of obesity as genetic markers cannot be applied and relevant characteristics of the peers' parents (such as PA of peers' parents) are not available in our data.

Table 5
IV estimates of peer effects on adolescent's BMI

Approach 1: BMI of peers' parents		
Variables	BMI (z-score)	
	LIML	GMM
	(1)	(2)
Panel A: First stage IV		
Peer's average mothers' BMI	0.068***	0.068***
SE	(0.009)	(0.009)
95% CI	[0.052, 0.085]	[0.050, 0.086]
Peer's average fathers' BMI	0.045***	0.045***
SE	(0.010)	(0.011)
95% CI	[0.025, 0.066]	[0.023, 0.067]
Panel B: Second stage IV		
Average peer BMI	-0.073	-0.074
SE	(0.126)	(0.113)
95% CI	[-0.320, 0.174]	[-0.296, 0.149]

¹² As a robustness check, we also use a weighted allele score as the instrument but again obtain similar results to those in Table 4. Following von Hinke et al. (2016), the weights for calculating weighted allele score are defined by the effect size of the obesity-related genetic variants based on an independent meta-analysis (see Speliotes et al., 2010).

<i>F</i> -statistic (global)	9.72	16.36
Under-identification test: Kleibergen-Paap rk LM statistic (<i>p</i> -value)	96.901(0.000)	59.984 (0.000)
Weak identification test: Wald <i>F</i> -statistic	55.211(>10%)	45.900 (>10%)
Over-identification test: Hansen <i>J</i> statistic (<i>p</i> -value)	0.164(0.686)	0.168 (0.682)
Controls	Yes	Yes
Country dummies	Yes	Yes
<i>N</i>	605	605
<i>R</i> ²	0.279	0.279
Approach 2: Mendelian Randomization		
Variables	BMI (z-score)	
LIML		
Panel A: First stage IV		
Unweighted allele score	0.052	
<i>SE</i>	(0.042)	
95% <i>CI</i>	[-0.030, 0.134]	
Panel B: Second stage IV		
Average peer BMI	0.761	
<i>SE</i>	(1.422)	
95% <i>CI</i>	[-2.037,3.558]	
<i>First stage F</i> -statistic	1.56	
Weak identification test: Wald <i>F</i> -statistic	1.559 (<10%)	
Country dummies	Yes	
Controls	Yes	
<i>N</i>	335	

Notes: The dependent variable is individual z-score of BMI based on 2012 IOTF criteria. The controls for the models in scenarios 1 and 2 are adolescent characteristics (age, gender, AVM consumption, meal frequency, health-related quality of life, number of friends and time spent together) and mother and family characteristics (age, occupation, BMI and household income). The instruments for the models in scenario 1 are peers' parental average BMI. The instrument for the model in scenario 2 is individual unweighted allele score. Robust standard errors are in parentheses (*SE*).

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

V. DISCUSSION AND CONCLUSIONS

To remedy the dearth of European empirical research on whether – and through which mechanisms – peer weight status and behaviour patterns might affect adolescent body fat (Salvy et al., 2012), this analysis uses I.Family Study data to probe for such effects on three different measures of overweight (BMI, waist circumference and body fat). Our results, derived for a sample of adolescents aged 12–16 from six European countries, provide evidence of peer effects irrespective of the measure of overweight, although the estimates for each of these do differ in magnitude. We also find that these effects are stronger among individuals at the upper end of the body fat distribution. This is a common finding in the literature (see, for instance, Trodgon et al., 2008; Halliday and Kwak, 2009; Loh and Li, 2013; Nie et al., 2015) and may – aside from capturing a peer effect – also suggest that adolescents with higher weight status are subject to greater stigmatisation and exclusion, and therefore tend to adopt similar behaviours (e.g. not being active outdoors or staying indoors at the computer/TV). However, as Trodgon et al. (2008) have highlighted, the results may also reflect selection being higher at the right end of the overweight distribution.

Turning our attention to possible pathways through which peers might influence adolescent overweight, we find evidence that both the adolescents' consumption of less healthy foods (SSBs, simple sugar, fatty foods, and fast foods as meals) and their YHEI are positively correlated with those of their peers. The elasticities¹³ of peer effects in SSBs, simple sugar, fatty foods and fast foods as meals are 0.180, 0.173, 0.230 and 0.187, respectively. In the case of fast foods, this would imply that, on average, an individual's fast food consumption would increase by 0.187% in response to a 1% increase in the peers' fast food consumption. These results are well in line with the elasticities for fast food in Fortin and Yazbeck (2015) and Ali et al. (2011b) for the U.S (0.208 and 0.178, respectively). We find no such association, however, for the consumption of healthy foods (e.g., vegetables, fruits and other fibre-rich foods). This observation is similar to that of Ali et al. (2011b) for the U.S., who also find no evidence of peer effects on fruits and vegetables. A positive association also exists between the time adolescents spend on leisure time PA and AVM and the time spent by their friends on similar activities, with elasticities of 0.268 and 0.108, respectively (in Ali et al., 2011b, in the elasticities for pursuing an active sport and regular exercise are 0.079 and 0.184, respectively). These findings suggest that peer effects on adolescent overweight could operate through friends' behaviour patterns, particularly unhealthy food consumption, physical activity and sedentary behaviours.

The strength of our analysis lies in its rich set of body composition/weight status data, which considerably extends the limited research evidence available for (continental) Europe, the range of behavioural measures included representing diet, physical activity and sedentary behaviours and the approach to analysis to accommodate endogeneity effects. Its main limitation is its cross-sectional design and relatively small sample size, which makes a causal analysis difficult. Hence, although we attempt to identify causality by applying an IV approach (using peers' parental BMI and genetic variants), identification problems remain, and our results must be treated as associations. Our cross sectional data also make it impossible to explore the dynamic relationship between peer effects and individual bodyweight over time. Moreover, regarding physical activity, we only employ a partial measure of physical activity (unstructured activity in leisure time) which may not reflect school-based peer relations or those that take place indoors (e.g. structured sport activities), although it could be argued that unstructured leisure time physical activity is most likely to be influenced by peer groups.

¹³ We calculate the elasticities at the sample means.

Keeping in mind these methodological shortcomings, our finding that mainly unhealthy dietary patterns and PA are positively correlated between adolescents and their peers could be used in interventions targeting the peers in addition to the individual adolescent or by addressing the composition of peer groups (O'Malley et al., 2014). Like some other studies (Dishion and Tipsord, 2010; Prinstein and Dodge, 2008), our findings indicate the existence of a “social multiplier effect” – not only for obesity, but also for unhealthy dietary patterns as well as physical activity and sedentary time. Such a multiplier effect has been largely attributed to in-group social norms, i.e., perceptions that prescribe or influence behaviour (Schultz et al., 2007). By also addressing peers, even small changes by some group members seem to be able to shift group norms on dietary behaviour or PA (Graham, 2008). Yet, what has been known and used in social norms interventions for smoking (Mercken et al., 2012), drinking (Balsa et al., 2011), promiscuous behaviour and other problematic teen behaviour (McAlaney et al., 2010) since decades, has only come to the attention of adolescent obesity research or policy-makers recently. It becomes clear that when designing effective public health policies and social marketing campaigns, peer influence and social networks should be taken into account. Failure to do so may underestimate the cost-effectiveness of obesity prevention and intervention programs.

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